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VETERINARY MILITARY HONOURS.

On the whole, in Western Europe at least, the present war has so far afforded veterinary surgeons fewer opportunities of distinction than most past wars. Nevertheless, Sir John French's list of officers who have distinguished themselves contains quite a number of veterinary names. We are glad to see so many—it proves that our Army colleagues are doing their work well, and it shows that their merit is recognised officially, which has not always been the case in the past. Some officers, we note, are commended for "gallant and distinguished service in the field"; and this, when we remember that comparatively few have yet had the chance of distinguishing themselves "in the field" is especially gratifying.

One other point should appeal to the whole profession, even those members who take least interest in military affairs. Several of the names mentioned are those of men who are already well known to us in a strictly veterinary capacity. In one way and another, they have proved their interest in the common work of our profession, and have helped to advance our scientific knowledge. We all knew them as good veterinary surgeons; it is very gratifying to hear that such men have also proved themselves good soldiers.

THE CENTRAL SOCIETY.

It will be seen from the report of this Society's last meeting, which we publish to-day, that its forthcoming meeting on Thursday evening next is of more than usual importance. All members of the profession in the Counties of London and Middlesex are invited to attend, with the object of discussing the War Office circular and the R.C.V.S. letter regarding the army veterinary question. Should time permit, this discussion will be followed by one upon influenza in horses.

Both subjects are well worth discussion by a large meeting of veterinary surgeons. Most men intending to help in any direction with the army scheme have arranged to do so already; but there is still time for new helpers to come in, and for existing arrangements to be improved. Both these objects can be effected at a large gathering of practitioners.

We need not emphasise the desirability of the subsequent discussion upon Equine Influenza—everyone realises the present importance of the disease. There ought to be a large meeting at Red Lion Square next Thursday.

STUDIES UPON THE ACTION OF IRON SALTS IN THE REGENERATION OF THE BLOOD.

Schminke discusses (*Münch. Mediz. Wochenschr.*) this subject. Van Noorden is of opinion that the iron salts circulating in the blood are not directly used in the formation of hæmoglobin, but that they exercise a strong irritant action upon the hæmatopoietic organs of the bone marrow, and that the result of this irritation is an improvement in the condition of the blood. This opinion is supported by Hoffmann and Müller, who, after administering iron salts to animals whose blood had been reduced, found the bone marrow much redder and richer in erythroblasts than that of control animals which received no iron salts.

Schminke has now carried out a number of experiments upon the subject; and has arrived at the result that the iron salts first increase the cellular constituents of the blood by irritation of the blood-forming organs, and that these newly formed cells, which are still poor in blood-colouring matter, do not increase in their proportion of hæmoglobin until later.—(*Munch. Tier. Woch.*)

SEDATIVE ACTION OF SODIUM IODIDE INJECTED IN SMALL DOSES.

Champion has published (*Presse Médicale*) his experiences on this subject. Sodium iodide, injected in small doses into the cellular or muscular tissue, is absorbed exceedingly well, and does not cause pain. It produces interesting therapeutic effects, and above all a suppression of pain and of certain spasmodic conditions.

Champion, in the human subject, injects only two centigrammes dissolved in two grammes of water. Sterilization is accomplished by heat. The salt should be pure and desiccated; if crystallised, it would be a little irritating. Very often one or two doses are sufficient; but there are no disadvantages in repeating them.

No general effect worthy of note is produced; but relief is obtained in inflammations, without causing either the arrest, localisation, or absorption of suppurations.

This treatment is indicated in certain cases of phlebitis and arthritis. Good results are also observed in certain cases dyspnea and emphysema.

Sometimes the action is immediate, and sometimes it takes place progressively in the course of a little time. The action is not specific, either as an anæsthetic or a stupefacient. Sodium iodide is a vaso-dilator, which perhaps, when its action is fixed upon the part where the morbid condition is localised, modifies it and corrects the spasm of the vessels.

Comparing the action of sodium iodide with that of morphia, the author points out that the iodide is not toxic, does not diminish the digestive activity, and, above all, does not cause cephalic congestion.—(*La Clinica Veterinaria.*)

A Case of Symblepharon in the Dog.

J. Schautyr records this case, the subject being a dog eleven months old. The animal came under treatment on account of a discharge from the left eye, which had begun suddenly a fortnight before. The lower lid of the affected eye was adherent to the eyeball and to the membrana nictitans for almost the whole of its extent, only about 1/8 to 1/5 inch breadth of the edge of the lid remaining free. The upper lid only showed a partial adhesion with the eyeball and the membrana nictitans at the internal corner of the eye.

The adhesions were separated almost without loss of blood by means of the blunt handle of a knife. The tissues forming the adhesion were reddish in colour, and were easily broken down.

Repetition of the adhesion was prevented by applying an ointment of cocaine and vaseline into the conjunctival sac for a few days. Schautyr thinks that the condition was probably caused by an irritating chemical agent penetrating into the conjunctival sac and destroying the superficial layer of its mucous membrane.—(*Zeitschr. f. wissentl. u. prakt. Veterinärmedicin.*)

W. R. C.

CENTRAL VETERINARY SOCIETY.

[National V.M.A. Southern Branch.]

A meeting was held at 10 Red Lion Square, London, W.C., on Thursday, 4th February. Mr. F. W. Willett, President, occupied the chair and the following Fellows signed the attendance book :—Messrs. W. N. Thompson, J. W. McIntosh, A. L. Wilson, G. H. Livesey, W. S. King, J. Willett, Col. C. Rutherford, Messrs. N. Almond, R. Bennett, J. Rowe, C. H. Sheather, G. Gordon, Profs. J. Macqueen, G. H. Wooldridge and Mr. Hugh A. MacCormack, Hon. Sec. Visitors, Messrs. A. C. Duncan, J. H. Bennett, and R. Spittal.

The Hon. Secretary announced that, as the prints of the minutes of the last meeting were only handed to him on his arrival at the meeting that evening, he had not had time to paste them into the minute book, their confirmation must be postponed until the next meeting.

Correspondence.

The Secretary announced that letters regretting their inability to be present had been received from Prof. Reynolds, Capt. J. C. Coleman, and Mr. S. H. Slocock. A communication was also announced from the Royal Sanitary Institute giving particulars of the twenty-sixth course of practical training for meat inspectors. It was ordered that the communication should lie on the table.

Appeal for Veterinary Surgeons for the New Armies.

The Secretary read the circular letter from the Royal College of Veterinary Surgeons and Army scale for Temporary Commissions. [These appeared in our issue of Jan. 30, pp. 404 and 412.]

Mr. McIntosh thought that all the Fellows were sufficiently patriotic to recognise the importance of the documents that had been read. For some time the profession had thought they did not receive from the State that recognition which it deserved, but the present was not the time to belabour that point. At this moment, when the very life of the country was at stake, all personal and private interests shrink into insignificance before the grave decision which war involves, and Fellows should do their very best to assist as members of the profession and as citizens of the Empire. There were some whose duties did not permit of their getting away very readily, and others were getting somewhat advanced in years, but he was quite sure that those who were not eligible for the field were anxious to do what they could at home. Personally, he should be very pleased to render all the assistance he could in conjunction with his own work, but further than that he could go. He supported the request of the College Council whole-heartedly, and thought the Society should do its best to assist in the matter.

Mr. Almond enquired whether any legal protection was likely to be afforded to a member going abroad whose practice was taken over temporarily by a neighbouring colleague. At a previous meeting when the subject was discussed, Sir John M'Fadyean stated that in Germany he thought some form of protection was given under the same circumstances, and it was desirable that something of the kind should, if possible, be adopted in this country. The College undoubtedly possessed a strong power in treating any divergence from an honourable course as disgraceful conduct, so that members would to a certain extent be protected in that way.

The President thought that when a member handed over his practice in the way suggested, the various parties would sign a letter, and that would constitute a legal protection on which action could subsequently be taken if there was any abuse of the agreement. No one would hand over his practice simply with a verbal arrangement. Should either practitioner die suddenly it might have serious consequences.

Mr. C. H. Sheather moved that copies of the communications from the Royal College of Veterinary Surgeons be sent to all the members of the College in the London and Middlesex districts on the Register, together with a short covering letter asking them to attend the next meeting of the Society in March when the matter would be discussed.

Mr. J. Willett, in seconding the motion, thought it would be much better if the practitioner who remained at home did the work for the one who went abroad and booked it as for him, leaving it to him when he came home to settle up the matter. He foresaw great difficulties in taking over clients, some of whom would probably run into debt, and against whom legal proceedings would have to be taken to enforce the payment of fees.

In reply to a question by Prof. Wooldridge, Mr. Willett said that in his opinion the man who held the practice in the first instance should render the accounts to the clients. It had been suggested that in the case of large country practices one or two practitioners should divide the working of that particular practice, one doing it one month and another the next month ; but the difficulty of that arrangement would be that some cases would probably be left over at the end of the month which the next practitioner would have to look after.

Mr. Almond thought that if Mr. Willett's plan were adopted the better course would be for the gentleman going away to furnish a list of his clients, and for the neighbouring practitioners to parcel it out amongst them.

Mr. Livesey, in seconding Mr. Sheather's proposal, said that of the veterinary surgeons in Brighton four had gone on service. He had offered to carry on his neighbours' practice so far as he could during their

absence and to satisfy their clients. He thought anyone who had any common decency would do his best for a fellow who had gone to the front, and would hand back the practice in its entirety. In such a profession the members surely should not be called upon to give a guarantee; it was a reflection on their own honour.

The veterinary surgeons in Brighton knew each other sufficiently well to know that they would not do such a thing as to try and grab a client from one who was serving his country. The population in Brighton was rather a movable one, and as there might be some difficulty in collecting fees he had always requested the clients to pay him personally for the work. He would hand over that money to the other practitioners when they returned. He thought it was rather hard lines to do work for a man in his absence and leave him to collect the money on his return, the collecting of the money being a jolly sight harder than doing the work.

Prof. MACQUEEN thought the whole thing was unnecessary, and that the War Office should look after themselves. There was nothing to prevent them making direct application to each practitioner on the Register, and if they offered better terms they would get plenty of men.

Mr. Sheather's resolution, seconded by Mr. Livesey, was then put and carried unanimously, and it was also unanimously agreed, on the motion of Mr. J. Willett, seconded by Mr. King, that a notification be sent to the War Office from the Society to the effect that they should approach the public bodies of the country with a view to their sparing the veterinary surgeons in their employ for the service of the country.

MORBID SPECIMENS.

The PRESIDENT exhibited two abnormally large lymphatic glands taken from the neck of a mongrel bull puppy ten weeks old. In reply to various questions that were asked, the President stated that the glands were taken out just below the larynx. There was no history as to the length of time they were forming. No other enlarged glands in the body could be felt externally.

Prof. WOOLDRIDGE promised to have sections made of the exhibit, and if anything interesting was discovered to report it to the Society.

NOMINATION.

Mr. R. SPITTAL, M.R.C.V.S., Rotherhithe, was nominated, and will come up for election at the next meeting.

SIDEBONE LAMENESS.

A ballot then took place for a subject for an impromptu discussion, and eventually the subject of sidebone lameness was chosen.

Prof. MACQUEEN, in opening the discussion, said the first statement he desired to make regarding sidebone lameness was that he was extremely sceptical of its existence, as he had never seen a case of pure sidebone lameness. Every case he had investigated where sidebone might appear to be a cause of lameness, revealed on further examination, some co-existing disease, and he had never been able to alter the view that he formed some years ago, that sidebone alone was not responsible for lameness. The reason he held that view might be stated very briefly as follows:—The ossification of the lateral cartilage was not usually produced by an injury; it usually took place very slowly, and he looked upon it as a physiological change which began a little sooner and extended a little faster in some horses than in others. In old horses the lateral cartilage of the foot, like all other cartilages of prolongation in the body, underwent a slowly extending ossification, and it would be found, he thought, that the lower border of the lateral cartilage in many old subjects was first engaged in that ossifying process. That was one reason why he did not believe that sidebone caused lameness except when complicated by some other disease.

Another was that half the lateral cartilage stood above the level of the hoof, and although this part of the cartilage might be ossified and quite inelastic he failed to see how it could produce lameness. It was necessary to consider whether the ossifying process was a painful one at any time or not, and he was unable to say that the transformation of the lateral cartilage, whether gradually or rapidly produced, was accompanied by pain. Those were the three principal reasons why he did not believe there was very often a case—he would not say never a case—of uncomplicated sidebone lameness.

He had also to meet what might be regarded as a different view, namely, that sidebone often co-existed with what was called contracted foot, and that if there were no sidebone there would be no contraction of the hoof. That he did not agree with, for the reason that in many contracted feet there was no sidebone. There was no alteration in the cartilage except towards its attachment on the os pedis. The supporters of the view he had just referred to sometimes claimed that, owing to the presence of an unyielding ossified cartilage, the hoof, when contracted squeezed the sensitive laminæ against the sidebone. He had no sort of faith in that view. The contraction itself was gradually produced, and the sensitive laminæ accommodated themselves to the condition. In connection with the view he had just mentioned it was desirable to consider the effect of the operation called hoof section. It was claimed that if there were no compression of the laminæ there would be no lameness, and assuming that the lameness was not due to compression of the laminæ the sawing of the hoof should not relieve the pain or lameness. That was perhaps a little difficult to explain, but he looked upon sawing the hoof in those cases of so-called contracted foot as a sort of counter-irritation. No horse was made sound by the operation until after the healing process had considerably advanced, and although the relief by section conferred a certain benefit it had only a temporary effect. He looked upon the treatment of ossified lateral cartilage, otherwise sidebone, as absolutely futile. He considered that anyone who nowadays fired and blistered an ossified cartilage was doing that which he ought not to do. The idea of line-firing, puncture-firing or blistering over an ossified structure such as a sidebone seemed to him ridiculous. There was no possibility of restoring the ossified cartilage to its former elastic condition; the blistering could only affect the upper half which was not exposed to compression, and which was not concerned very much with movement within the foot. He went the length of saying that the treatment of sidebone by counter-irritation had no sort of justification. Those lamenesses which were commonly attributed to sidebones were cases of lameness due usually to disease of the bones, the os pedis, os corona, or possibly the navicular, and in other cases not connected with disease of bone there was usually some chronic disease of the secreting membrane of the hoof.

Mr. ALMOND said no one would deny that sidebones and lameness were frequently found associated, but it required a certain amount of conviction to conclude that sidebones were the cause of lameness. It was no doubt the experience of most of the Fellows that horses frequently had sidebones that were not associated with lameness, and therefore when lameness was found associated with them it was natural to look for other evidence as a cause for the functional disturbance. The opener had stated that sidebones uncomplicated did not give rise to lameness, but he clearly indicated that sidebones themselves were really insensitive structures, and the ossifying process was slow and not of an inflammatory nature. To that extent he agreed with

him. Prof. Macqueen did not indicate very clearly the nature of the complications which led to lameness as associated with sidebones, but he conveyed the impression that compression of the sensitive structures between the sidebones and the hoof was not likely to give rise to lameness. Personally, he did not take that view, as he thought that where a bulging of the hoof over the seat of the sidebone was obtained, although it took place very slowly, it had existed for a considerable time, and it was only when it arrived at a certain period that lameness arose. Prof. Macqueen stated that section of the hoof over the seat of the sidebone gave only temporary relief. That seemed to him evidence of undue pressure upon sensitive structures in the hoof, which pressure was relieved by the section. In his opinion that proved that the lameness was caused not by the sidebone itself, but by the compression of the sensitive structures between the sidebone and the hoof. He did not regard sidebone as a cause of contracted feet, and had never heard such a suggestion made before. Many young horses had sidebones with very wide open feet, and good, well-developed frogs. In such a foot he would not expect the horse to go lame, and he never hesitated to recommend a client to buy a horse whose feet bore that description. He had one or two clients who preferred buying a horse with sidebones.

If sidebones did not cause contracted feet what did? In his opinion the cause of contracted feet was the absence of use, the absence of frog pressure, the failure to bring the due weight on to the heels of the foot, *i.e.*, contraction followed lameness, or tenderness, or inability of some kind. The frog was a natural expanding material for keeping the heels open, and it was the want of the use of the frog, which might be due to its being raised off the ground or due to lameness, which gave rise to contraction of the feet. In the treatment of sidebones, he agreed with the author as to the want of logic in applying irritating substances with a view to affecting insensitive structures. That would hardly be the reason for applying a blister or other irritating substance to the coronet. He did not think he would ever do such a thing himself for that purpose, but if he did apply a blister, the explanation would be to stimulate the horn-secreting membrane to produce a rapid and comparatively yielding horn, so that the bulging might be rendered more easy, thus facilitating the expansion of the hoof, which, by section, another form of treatment, frequently gave rise to relief. Many years ago, before the section of the horn was brought into use, it was his practice to thin the horn as much as possible over the sidebone with a view to allowing the expansion or bulging which might give relief, and in many cases that result followed. The patients in those cases were chiefly farm horses, doing slow work on soft ground. He found that a useful practice, although he did not think it was at all equal to vertical section of the horn.

Mr. J. WILLETT said he joined issue with the opener's statement as to sidebones causing lameness. In his experience of the bulging of the horn in the region of sidebones on making a section he found that the horn in that particular part was much thicker than normal, and when a sidebone was well developed there was no doubt a great deal of undue pressure from the horn itself. Great relief was obtained by thinning the horn over the region of the sidebone, or by making V-shaped grooves with a hot iron or with a gouge. In a horse which was supposed to be going lame from sidebone, if a bar shoe was put on, the pressure on the bearing surface over that region was relieved, or a bar pad might be used, preventing the concussion, and it was frequently found that the horse worked much better. For that reason he could not agree that sidebone did not cause lameness in that particular case.

Mr. MCINTOSH said that for a long time he looked upon sidebone as a somewhat serious defect. His experience of a stud of 900 horses had altered his views, and he was now inclined to agree with Prof. Macqueen —that sidebone, when fully formed, very rarely, if ever, caused lameness. He preferred a horse without a sidebone, but as it was impossible to find all horses absolutely clean he acted on a rule, that if the sidebone was well clear of the coronary band, there was little fear of trouble arising from the defect; and following these animals up, as he was able to do, he had not found any trouble arising from them.

He thought it possible that sidebone, during formation, might give rise to pain, and as a result, lameness, and there were times when it was extremely useful to be able to attach lameness to something.

At this stage he was of opinion that the application of a blister was of some value, and although it could not possibly remove the sidebone, it helped to hasten the inflammatory process to its final issue.

He did not think there was any connection between contracted feet and sidebones. It was true we occasionally found them existing together, but he thought more frequently the one without the other. He believed, as the opener had stated, that in contracted feet the sensitive structures accommodated themselves to the altering condition of the horn, and unless associated with some other disease the condition itself never caused lameness.

Mr. THOMPSON agreed with Prof. Macqueen that an uncomplicated sidebone did not cause lameness, but it was often very hard to say when a sidebone was complicated and when it was not complicated. In my post-mortems it was found that the inside of the process of the os pedis was very much roughened, and ostitis had existed on the inside of the bone. In some of those cases lameness undoubtedly occurred. Where bulging of the foot was obtained he did not think it could be called an uncomplicated sidebone, because evidently something more than the ossification of the lateral cartilage existed. In some of those cases he believed Smith's operation did good. He had known some very big cart horses work sound for a long while after that operation. He did not think he had ever been actually able to trace lameness to an uncomplicated sidebone. Many cases of lameness existed in which there were also thickened tendons, sidebone and ringbone, but none of them were the cause of the lameness, which was probably due to a picked-up nail or something of that kind. It was often very hard to say what was the actual cause of the lameness.

Putting on one side congenital contraction, he did not think contraction of the foot as a primary lesion existed. A strained tendon, a ringbone or any other trouble which caused an animal to go on the toe caused a contracted foot. If a horse was shod with a bar shoe long enough a contracted foot would result. In all cases, as far as he could judge, it was a question of cause and effect. If they took away from the frog its natural anti-concussion and anti-slipping functions atrophy of the frog would result, the walls fell in and a contracted foot was obtained. That was proved by the fact that if when a contraction of the foot was obtained without any complications, the horse was shoed with tips the wall of the foot would go back to the normal position again. He agreed with Prof. Macqueen in his suggested new treatment of sidebones, as there was no lameness there was no need for treatment. In years gone by when he was in general practice farmers frequently brought him horses and said they had sidebones and asked him to fire the animal. It would certainly not have encouraged the ledger account if he had replied that he would not fire him, the farmer would probably have taken him to somebody else.

Under those circumstances, he puncture-fired the animals.

Mr. WILSON agreed with Prof. Macqueen that sidebones did not cause lameness. His company had about a thousand horses, some of which were bought with small sidebones, and a very large percentage of them acquired sidebones during the six years they were in service. He did not call to remembrance a single case in which sidebone had caused lameness. He therefore did not adopt any treatment, not having a ledger account to worry about.

Prof. G. H. WOOLDRIDGE said that probably he started out from his surgery classes prejudiced with the view that Prof. Macqueen had put forward. On the other hand, he had exercised his powers of observation for a considerable number of years since, and he had examined, very carefully and on numerous occasions, horses affected with sidebones where lameness was present, and those observations had led him to accept the teaching he had previously received as being correct. He had seen no reason whatever to differ in any sense from the views that had been expressed by Prof. Macqueen in opening the discussion. He did not think Mr. Almond said quite what he meant in one respect, when he referred to bulging of the hoof in sidebones down the front of the toe. Personally he had never yet seen a case of bulging of the hoof at the toe due to sidebone. In cases where such bulging had occurred it had mostly been due either to some disease of the pyramidal process of the os pedis or of the lower portion of the os corona. He did not think the squeezing of the sensitive lamimæ in sidebone was a possible cause of lameness. The ossified cartilage did not yield at all; the bone was not pressed outwards; there was no pinching of the sensitive laminæ covering the lower portion of the ossified cartilage, and consequently he could not see how that could exist as a cause of lameness. In other cases where lameness existed in the presence of sidebone, on careful examination it was sometimes possible to appreciate the presence of exostosis on the posterior external margin of the os corona or the os suffraginis. The lower seat was rather difficult to feel if there was a well-marked sidebone, because the sidebone rather covered it, and it was difficult to feel down behind, but in a number of instances where he had boiled out feet where he was not able to detect the presence of exostosis of the coronet bone he had been able to find it subsequently. In the majority of cases where the feet were contracted the presence of navicular disease was discovered, so that although one might at the time of examination be unable to find the other cause, *i.e.*, the actual cause of the lameness, he was quite certain there was another cause in operation. As Mr. McIntosh had said it was necessary sometimes to attach the lameness to something, and the practitioner therefore took refuge behind what appeared to be the obvious thing. It reminded him of a statement he heard Mr. T. S. Price make on one occasion with reference to rheumatism. "Some people," Mr. Price remarked, "said that rheumatism as a distinct disease did not exist, but personally he had carried the name about with him and found it useful for forty years; it had stood him in good stead, and he intended to stick to it." For the same reason some of the members might stick to the idea that sidebone caused lameness. The explanation of the relief that followed hoof section or hoof grooving in cases where sidebone existed was, he thought, the fact that the co-existent condition, which could not always be discovered, had been benefited by the temporary rest and the counter-irritant applied, and that the latter was thought to have been beneficial to the actual sidebones.

Mr. R. BENNETT agreed with Prof. Macqueen's statement that uncomplicated sidebone did not cause lameness. This opinion was no doubt influenced largely by very much the same reason as that given by Prof. Wooldridge, namely, that he started out with that idea when he first entered on his professional life. One question had not been raised which he had not been able to settle in his own mind, namely, was a foot with a sidebone more likely to develop ostitis of the pedal bone or of the os corona than a foot that had no sidebone? Did the ossification of the sidebone tend to spread to the other structures of the foot, or was the sidebone a sequel to the ostitis of the pedal bone?

Mr. C. H. SHEATHER said he entirely agreed with what Prof. Macqueen had said with regard to sidebone *per se* causing lameness. He thought in order to discover whether lameness existed, provided there was a sidebone, it was necessary to look on the inside and the outside of the sidebone, a fact which Prof. Wooldridge had rather overlooked. He had noticed in a great many cases where prominent sidebone existed that in viewing a horse from a going away position, when he turned round he limped, but when he came towards the observer he did not look so bad. That was due, he thought, to the nipping of the sensitive structures between the os corona and the sidebone, and he was rather inclined to think that similar nipping occurred on the outside. If a horse had good open feet a little hardening of the cartilage of a sidebone, but nothing on the pastern, he would buy him every time, but if he had anything on the pastern he would not do so. He thought the structures on the inside were more important than those on the outside of the sidebone.

Mr. ALMOND enquired whether Prof. Macqueen attached more importance to a single sidebone than to a double sidebone in a horse's foot.

Mr. J. WILLETT, in support of the argument he had previously adduced, mentioned the case of a horse he saw the previous day, which came in from a field-day quite lame. He had two very well developed sidebones on one foot, and the horn over the region of the sidebone was thick. He had the horn thinned well down, put on a cold swab, and the horse was now nearly sound. He could find nothing else the matter with the leg; it was perfectly clean, with the exception of the sidebones mentioned.

The PRESIDENT said that he remembered as a boy a roan mare on a particular farm having four or five colts, all of which developed sidebone at three or four years old. His father had the walls thinned: made three or four fissures with a firing iron and blistered them, and every one of the animals went sound and worked for many years, although they had previously been lame.

Prof. MACQUEEN, in reply, said he had very little to reply to, as the Fellows had answered one another very satisfactorily. The only speaker who seemed at all inclined to object to his opening observations was Mr. Willett. He desired to impress on them the fact that, before coming to a conclusion regarding sidebone as a cause of lameness, it was necessary to consider why, if sidebone caused lameness, every sidebone did not cause lameness. Everyone was willing to confess that a very large number of horses with conspicuous sidebone were not lame. Why? What was the change that must be in operation in those exceptional cases which were claimed by Mr. Willett as examples of sidebone lameness? Was it sidebone alone or sidebone *plus* something which Mr. Willett had not been able to diagnose? Mr. Willett had stated that as a result of his experience the thing bulged. What was it that bulged? The hoof? Was it the sensitive membrane? Was it the sidebone? In his opinion, Mr. Willett's observation was at fault, and he did not actually know what bulged, if anything bulged at all. Mr. Willett had stated that in the case he had examined on the previous day the hoof was enormously thickened. Why? A hoof could not be thickened from within outwards by sidebone. The thickening of the hoof depended upon the secreting

surface, and if that was extended a thick hoof resulted But that had nothing to do with sidebone. The horse that Mr. Willett relieved by thinning the wall over the sidebone had possibly had a bruised coronet, or had possibly suffered from some other injury not apparent on the outside. One was very apt to fly to conclusions, and it was one of the weaknesses of veterinary practice, so far as diagnosis was concerned, that the average practitioner in his haste often selected the most obvious cause as the easiest way out of the difficulty. A horse was brought to him and he found a sidebone; he assumed that his employer would probably accept that as a sufficient cause of lameness; and if the veterinary surgeon was in a hurry he made no further effort to ascertain what the real cause of the lameness was. He asked those who adopted that view how they reconciled the fact that the majority of horses with apparent sidebone were not lame, with the further fact which some practitioners claimed—that a few horses with sidebone were lame? What made the difference? Was it in the sidebone or was it in something else? Personally, he said it was in something else.

A very pertinent remark had been made by Mr. Bennett which he put in the form of a semi-question, namely, whether the existence of a sidebone predisposed a horse to ostitis of the os pedis or of some other similar disease. That was one of the chief objections to the breeding of horses with sidebone. Sidebone was an index to something that was far worse in many instances, fortunately not in all. Many practitioners recommended the purchase of horses with sidebone affected feet which were of good shape, and rejected horses with sidebone feet which were of bad shape. The explanation was that in certain of those cases sidebone should be taken as indicating the probable occurrence of ostitis of the os pedis. In the cases referred to by Mr. Almond where he believed that there was bulging of the wall, that bulging was not, in his opinion, produced by sidebone immediately; it was produced, if it existed, by ostitis of the os pedis. Sometimes there was bulging of the unossified lateral cartilage as an effect of ostitis of the os corona, because the os corona stood within a portion of the lateral cartilage; and so long as the lateral cartilage was resilient it could be bent or bulged by the exostosis or outgrowth of bone which formed beneath it.

He thought Mr. Bennett's statement was one of considerable importance. There was no doubt a danger of ostitis of the os pedis as a possible complication of sidebone in a foot of indifferent shape. Mr. Almond asked whether he considered a one-sided sidebone was better than a double sidebone. He did not look upon the one condition as of any more importance than the other; he thought they were both harmless so long as the feet were of good shape. In feet of bad shape there was greater danger of the occurrence of ostitis, not necessarily owing to sidebone, but to defective bone structure transmitted from parent to offspring or acquired, in consequence of hard work. He agreed entirely with what Mr. Thompson said regarding contracted feet. In his opinion contracted foot was a primary condition only when it was congenital. He had not the slightest doubt that horses were born with abnormally narrow feet, and they remained so throughout life, but there was a contraction which could be acquired very readily, in fact hastened in certain cases by the treatment adopted. That form of contraction was due, as Mr. Thompson explained, to loss of function of the ground surface of the foot, particularly the heels and frog.

On the motion of Mr. J. Willett, seconded by Mr. McIntosh, a hearty vote of thanks was accorded Prof. Macqueen for opening the impromptu discussion; and, on the motion of Prof. Wooldridge, a vote of thanks was also accorded to the President for exhibiting his morbid specimen.

It was suggested that at the March meeting of the Society, if time permitted after the discussion of the communication from the Royal College of Veterinary Surgeons in reference to the supply of veterinary surgeons for the new Armies, a discussion should be held on "Influenza in the Horse."

HUGH A. MACCORMACK, Hon. Sec.

THE VETERINARY MEDICAL ASSOCIATION OF IRELAND.

(NATIONAL V.M.A.—IRISH BRANCH).

A general meeting was held on Wednesday evening, 25th November, 1914, in the Gresham Hotel, Upper Sackville Street. Mr. J. B. Dunlop in the chair. There were also present, Messrs. Howard, McShane, Ebbitt, Purcell, Watson, Wilkinson, Prentice, Dolan, McCann, Magee, and Profs. Craig and O'Connor.

Apologies were received from Messrs. Reavy, Heney, Winter, Chambers, Patrick, and McKenny.

The minutes of former meeting, which had been circulated, were adopted on the motion of Mr. Watson, seconded by Prof. O'Connor.

The HON. SECRETARY read the following report of Council:—

"A meeting of the Council was held on Wednesday, Nov. 4th, 1914. Letters were received from other Veterinary Associations giving their methods of securing publication of the proceedings of their meetings simultaneously in the two Veterinary Journals, viz., *The Veterinary News* and *The Veterinary Record*, the usual method being to prepare two copies and send one to each. The Hon. Secretary was instructed to make arrangements with the reporter to supply two complete copies of the report of each meeting and send one to each journal. Resignation of membership was received from Capt. Tate. The Secretary was instructed to write Capt. Tate and ask him to continue in the Association as a corresponding member at an annual subscription of 2/6.

A letter was received from Mrs. Mettam asking for a subscription to the fund which she is collecting for Mrs. Moore to send comforts out to the men of the Army Veterinary Corps. The Council recommend that a subscription of two guineas be made.

The Hon. Secretary announced that Mr. W. P. Power had gained the medal given by the Association to the student in the Royal Veterinary College of Ireland who obtained the highest aggregate of marks in the July professional examination of final year students."

The report of Council was approved.

Mr. WATSON: I think it is most desirable that we should support the Fund for the A.V. Corps. I would like to know is it competent to give the money out of the funds?

The CHAIRMAN: It is a question whether it should be that or a matter of private subscription.

Prof. O'CONNOR: You can do both.

The CHAIRMAN: There is no objection to both. Most of the members have subscribed themselves.

Mr. WATSON: I think there are certain conditions laid down for dealing with the funds. I for one would be delighted if we gave £10. I think that the funds are for certain purposes laid down in the minutes.

Mr. HOWARD: This Association has in the past voted small sums of money for such purposes and no question was raised as to whether we were doing what was proper. At the Council meeting it was suggested that two guineas was not a bad subscription considering that the members have subscribed.

Mr. WATSON: I have no objection to giving £10 but I think the money is earmarked for certain purposes. Perhaps the Treasurer can tell us.

Prof. CRAIG: So far as I am aware the only disbursement I can make is at the request of the Association. That is the only instruction given to me.

Prof. O'CONNOR: There is a rule which states "No gratuitous disbursement shall be made of the funds of the Association until same shall have been considered by the Council and approved by the Association."

Mr. HOWARD: It has been considered by the Council and the Council made a recommendation to give the sum of two guineas.

The CHAIRMAN: That is admitted, but Mr. Watson wants to know if it is in order.

Prof. O'CONNOR: This rule puts you in order.

Mr. WATSON: I do not think it applies to my question. I am looking for light and leading.

Mr. HOWARD: I think the rule is very clear on the matter.

Mr. HOWARD: I formally propose that the recommendation of the Council be accepted.

Mr. WATSON: I beg to second it and that the amount be increased from two guineas to five guineas.

Mr. PRENTICE: Perhaps Prof. Craig would tell us how we stand.

Prof. CRAIG: So far as the funds are concerned there is a credit of £40. I do not know what the disbursements will be before the end of the year.

Mr. WATSON: £40 is a good balance and £2 2s. is a miserable sum to give.

The CHAIRMAN: I quite agree £5 5s. seems reasonable. What do you say Prof. O'Connor?

Prof. O'CONNOR: Five guineas. The Central gave five guineas and I don't see why we should not.

Mr. WILKINSON: I would make it five guineas.

Mr. HOWARD: I am only too happy to make it five guineas.

The motion was passed, the subscription being unanimously five guineas.

New Member. Mr. D. J. O'BYRNE, Tipperary, who had been proposed for membership of the Association by Mr. McKenny and seconded by Prof. O'Connor, was unanimously elected.

Annual Dinner. Prof. O'CONNOR: The question is whether we shall have the annual dinner under the present circumstances?

The CHAIRMAN: A great many men are away on military duty. I thing it would be better to postpone it.

Prof. O'CONNOR: I think it should be postponed. I do not think we would have a big gathering, and I do not think it is a time for dinners.

Mr. Prentice and Mr. Howard concurred.

It was unanimously decided that the annual dinner be not held this year.

THROMBOSIS OF THE ANTERIOR MESENTERIC ARTERY OF THE HORSE.

By L. M. MAGEE, M.R.C.V.S., Dunboyne.

Mr. Chairman and Gentlemen,

I do not think it necessary to apologise for the choice of subject which I have made for discussion to-night. With the exception of three cases which I recorded about two years ago in a paper on "Some successful and unsuccessful clinical cases," as far as I know there has been no mention of the disease at any of our meetings during the thirteen years I have been a member of the Veterinary Medical Association of Ireland. I can, therefore, safely claim that the subject is at least not hackneyed.

When one becomes very interetsed in a particular disease there is a tendency, perhaps, to get it on the brain, as they say, and there may, as the result of one's enthusiasm, be an inclination to attach undue importance to it; that may be my case with regard to thrombosis of the anterior mesenteric artery.

I don't think I am exaggerating this importance when I say that not only does this pest cause the loss of a considerable number of aged animals, but it accounts for the death of more young horses in these countries than any other disease. That may seem a tall order: I am speaking however from my own clinical experience. I thought of referring also to some of those conditions in which other arteries are affected by thrombosis, such as the posteria aorta and iliacs, but as I have not, at least knowingly, come in contact with this affection in other blood vessels, I decided that I would only bring forward that phase of the disease on the clinical aspect of which I could speak from personal acquaintance. Such a course is more likely to develop an effort to break new ground; it makes an essay more interesting, and tends to stimulate a lively and profitable discussion.

I do not think we fully appreciate the seriousness of this disease, nor the losses it entails.

Like the average practitioner, what appeals to me most is its clinical side, and I intend to direct most attention along that line. I am not about to suggest a remedy, nor can I announce any very startling discovery, for the scientific investigation of those obscure portions of the life history of the little worm that undoubtedly causes the thrombus is hardly within the domain of the veterinary surgeon in private practice; still I may, perhaps, be able to add a little to our clinical interest in the disease.

With a thorough knowledge of the life cycle of the parasite, we may be able to prevent its ravages or the frequency of its attacks, but once the disease is established and has destroyed the function of a vital artery, from its nature I am afraid it will always be a case of shaking our heads and looking wise. I have reason to believe that the disease, the symptoms of which are at times so obscure and so dissimilar in individual cases, is often not diagnosed, and I confess that I have often been at fault myself in that respect. If, therefore, these notes should play some little part in saving others from making my mistakes I think I will have done some service to my profession. We know that one often gets as much credit for diagnosing a fatal case correctly as for bringing about a brilliant recovery.

Thrombosis of the mesenteric arteries is almost invariably due to inflammation and obstruction caused by the larvæ of Sclerostomum vulgare (Loos) or Bidentatum (Sticker).

History. It will be sufficient to say that the worms were observed as far back as 1665, and have since been studied by different investigators, but there are still some very important points in their life history yet unsolved.

Occurrence. Bollinger has proved that over 90 per cent. of all horses, excepting foals, have a verminous aneurism. He also says that one half of all fatal cases of colic are caused by thrombosis or aneurism, but this is, no doubt, too high a percentage. The clinics of the veterinary colleges of Dresden, Berlin and Budapest, and the returns of the Prussian army, show an average of about 8 per cent, but it should be remembered that very probably this deals almost entirely with aged horses; if young horses were included, I am confident the percentage would be very much higher. The probability is that the disease is more common in some countries than in others, and in Ireland, for instance, perhaps even one county may be infested more than another. The disease also attacks foals, and a man named Poppel found in a foal ten days old an aneurism of the anterior mesenteric artery as big as a pigeon's egg, and in it a thrombus containing the larvæ of sclerostomum. I feel confident that the frequency of its occurrence in foals is not appreciated.

Etiology. The adult worm is from an inch to two inches long, brown or light grey in colour. They are found in the intestines, most commonly the large bowel,

adhering to the wall from which they suck blood, often causing a considerable amount of anæmia.

The immature worms or larvæ are about half-an-inch long, pale white in colour, and they select certain arteries as their habitat. The specimens of thrombosed mesenteric arteries which are before you on the table I obtained post-mortem from my last two cases of the disease; they contain the larvæ in great numbers.

The mode of their emigration into the intestines and from there into the arteries is not known for certain. According to authorities quoted by Hutyra and Marek, the ova of the mature worm inhabiting the large intestines are voided with the fæces, and from these free ova the larvæ are liberated in water, on moist walls of stables and in manure. From here they get again into the horse with drinking water, with food contaminated with manure, or with the grass of marshy pastures. Personally I don't think it is at all necessary for the pasture to be marshy or inferior. I have seen three cases occur on a stud farm that contains some of the most fashionable blood in the United Kingdom, where neither money nor trouble is spared in its management, and the land is the very best; the drinking troughs in the paddocks are so arranged that there is always a fresh supply of water that cannot be contaminated by the animals' fæces, and there is no stagnant water to drink from. In these instances, as probably in most other, it was very likely the grass that became contaminated. If this is so it looks as if there will always be great difficulty in preventing infection, unless we can prohibit the introduction of infested animals, which, in the present state of our knowledge, seems almost impossible. This would especially apply to such institutions as stud farms, where brood mares from different places are coming and going each season, and may carry the worms in their alimentary tract. It is said that after entering the intestines the larvæ later on bore into the wall of the bowel, and by getting into some of the blood vessels make their way in the blood stream to the anterior mesenteric artery, which is evidently what we call a predilection seat.

Pathogenesis. The larvæ adhere to the intima of the artery or bore into it. This causes inflammation and the formation of blood coagula, and the vessel itself becomes thickened and dilated. The gradual formation of the thrombus leads to a narrowing of the lumen of the artery, which may eventually become completely closed. This inevitably causes anæmic necrosis of that part of the bowel of which the blood supply cannot be restored by collateral circulation. I can, perhaps, better explain this by referring you to the drawing which I have made, which is an enlargement of a splendid illustration by Ellenberger and Baum, showing the anterior and posterior mesenteric arteries and their relations to the intestines. (The essayist then briefly described the anatomy of the parts and pointed out the most common seats of the disease in the arteries).

I will now quote two paragraphs on the disease from Hutyra and Marek, because they explain ideas which I wish to express, and in language better than I could command. They say: "A very slowly progressing narrowing of the lumen of larger, or of numerous smaller arteries, does not necessarily produce particularly grave disturbances, because the anastomoses and eventually the partly obstructed vessel have time to gradually dilate. However, complete closure of the trunk of the anterior mesenteric artery (*a*), of the ileo-cæcal (*b*), of the two arteries of the colon (*c*, *c'*), or of the arteries of the cæcum near their places of origin (*d*, *d*,), also obstruction of several neighbouring arteries of the small intestines (*e'*, *e'*) will cause irreparable disturbances of circulation, even if taking place gradually."

The second paragraph is: "The intestinal wall which is damaged in its nutrition, or eventually becomes necrotic through grave circulatory disturbances, early permits intestinal bacteria to enter the peritoneal cavity; these then multiply rapidly in a serous exudate and produce circumscribed, later general peritonitis, which causes continuous inflammatory pain; the bacteria and their toxins are also absorbed into the blood, and general infection or intoxication with their symptoms follow."

Anatomical changes. All cases where the circulation of the affected bowel cannot be restored terminate fatally. On post-mortem examination the parts affected are often found to be dark red or black in colour, and sometimes the wall of the bowel is greatly thickened. In one instance I found the wall of the whole cæcum blue-black in colour and nearly two inches thick. On the other hand I have had cases where the wall of the affected bowel was hardly discoloured or thickened at all. The thrombus in the artery is composed principally of fibrin in which the larvæ are scattered, sometimes in great numbers.

At times I have found the centre of the thrombus pierced by a narrow passage—the remaining vestige of the lumen of the artery—and containing a recent blood clot; through this the blood had evidently been feebly circulating before stoppage finally took place.

Symptoms. I consider that no particular set of symptoms can be said to be diagnostic. There is not even one symptom that I have found common to all the cases I have seen.

In aged animals an attack often sets in suddenly, with manifestations of colic for which there is no apparent cause. The peristaltic sounds may at first be even exaggerated and the bowels and kidneys working normally. On the other hand, peristalsis may be absent, and the patient may not have had a motion for a day or two. The attack may proceed like an ordinary case of colic and, in fatal cases, terminate in a few days. The temperature is nearly always elevated.

In mild cases recovery, of course, takes place. These are generally like a sub-acute attack of colic, which may only last an hour or so; in such instances the thrombus has probably only occluded, or partly occluded, the lumen of the artery and the blood supply can be restored by collateral circulation. The pain is due the temporary anæmia of the intestine, and is probably something like the sensation we call "pins and needles," which we experience sometimes when we cross one knee on the other while sitting, causing temporary compression of the popliteal artery at the back of the upper knee.

In fatal cases the fæces are said at times to be mixed with blood, but I have never observed it.

An attack may terminate in a day or two, or the animal may be noticed to be ailing for weeks, or even months, before it finally succumbs.

There is one symptom which to my mind, when present, and especially if accompanied by manifestations of pain, is almost diagnostic; but, strange to say, I have never seen it mentioned in any literature on the subject; it is the occurrence, without any apparent cause, either of partial paralysis of the hind legs, such as in ricked back, or of a complete inability of the animal to rise from the ground, almost as if the back were broken; there is, however, always sensation in the hind extremities, and the tail is not flaccid, which prevents it being confounded with spinal trouble.

Perhaps I can best present a clinical picture of the great variety of symptoms which may be observed in different cases by briefly describing some of my experiences.

Case I. I believe the first time I saw the condition was when acting as hospital surgeon in the Dublin Veterinary College, thirteen years ago. An aged cart horse was admitted to the College, apparently suffering from acute colic, and a thorough examination did not afford any information as to the cause. Treatment gave

no relief, and on the following morning, as the case was evidently hopeless, I got the consent of the owner to perform an exploratory laparatomy. Having chloroformed the horse and taken the necessary precautions, I opened at the left flank. It was an interesting but tiresome experience. After roaming about for over an hour through the abdomen, and having had a hand-shaking acquaintance with nearly every organ in it, my suspicion of a possible calculus, intussusception or twist ended. Eventually I pulled out a portion of the small intestine, some two or three feet of which were somewhat thickened and perfectly black in colour. I slit the bowel lengthwise for a few inches but obtained no information. Having suturned and returned the intestine I stitched the wound at the flank and allowed the animal to come to. He died during the night. A A post-mortem next morning threw no further light on the mystery, and all I knew was that the bowel was necrosed for some reason which I did not understand.

This was certainly a case of thrombosis, probably of several of the arteries going to the small intestine (*a*, *b*, *c*).

Case II. I had my next experience in private practice. It was an aged horse belonging to a carman. He had left the animal in the stall the previous night apparently in perfect health. In the morning the horse was found lying on its off side, unable to make any attempt to rise. At times there were slight attacks of dull pain; the bowels and kidneys were working normally; after a while both hind legs became swollen.

I forget what my diagnosis was, but I remember being far from happy as to the real nature of the case. The horse died the following day, but I was unable to make a post-mortem examination. Even had I done so I would not have looked for thrombosis, which I am confident was the cause of death.

Cases III. and IV. My next cases were two draught colts, not long weaned, on the same farm, about a month's interval between them. Their symptoms were very much alike. Nothing had been noticed amiss with either of them till they were found lying in the field unable to rise, and in pain.

When I was called to the first foal, he was *in extremis*, and with the owner's consent I put him out of pain by pithing. *Post-mortem*, with the exception of clumps of bots in the stomach, I discovered nothing of any interest. On account of his peculiar stiff appearance before death I suspected tetanus, and thought that possibly infection had entered through the wounds in the stomach made by the bots.

In the case of the second foal I suspected thrombosis, and tried to confirm my diagnosis by examination per rectum, which was not satisfactory. I arranged to have the carcase brought to the Veterinary College, but, owing to a misunderstanding, it was not sent for till rotten, and there was no *post-mortem*.

These three cases were in County Wicklow; the next was in Meath, and was one of those I recorded two years ago.

Case V. It was a foal of the same age and class as the previous two, and the history and symptoms were almost identical with those of the Wicklow foals. I made a confident diagnosis of thrombosis, but the owner scouted such a new-fangled idea, and insisted that the foal's back was injured, an opinion in which he was supported by all the "knowledgeable" men of the neighbourhood, who had assembled. A post-mortem two days later revealed a huge thrombus, containing great numbers of larvæ, which completely occluded the mesenteric artery. On demonstrating the thrombus and the little worms to the owner, he was good enough to acknowledge his mistake, and, by way of a *solatium* exclaimed, "Be gob, you're a rattler."

Case VI. The next was a two-year-old thoroughbred colt that I had castrated the previous season. While running at grass with others he was noticed by the herd to be "wobbly" on his hind legs, looking unhappy, and not feeding. He was immediately brought home, a distance of five or six miles, and was very much worse at the end of the journey. On my arrival shortly afterwards I found him down, unable to rise, and in great pain. It was a typical case, and I was confident of my diagnosis. He died next day, and the *post mortem* was confirmatory. The groom in attendance told me that before death the poor brute often screamed almost like a human being in agony.

Case VII. An almost identical case to number *II.* was that of an aged mare used for drawing a bread van. She was found in her stable early on a Sunday morning lying prostrate, in great pain, and unable to rise. After a thorough examination my mind was a blank. I eventually made a diagnosis of probable twist, though I was at a loss to explain to myself how this could prevent her from getting on her feet. Large doses of chloral hydrate, etc., gave no relief, and she died that night. I made a careful *post-mortem* next morning, but could find absolutely nothing abnormal; I never thought of examining the anterior mesenteric artery.

Case VIII. Another peculiar case was that of a seven-months-old half-bred filly foal. She was observed not to be thriving, was walking stiffly behind and feeding badly. She was brought in and drenched three times a day with gruel, milk and eggs, but as she did not improve, my advice was asked. She had not lain down since she came in, and stood in one position taking no interest in anything. Both hind legs were swollen and a thick œdematous swelling extended along the abdomen to the fore legs. She never showed any symptoms of pain.

An examination per rectum was again disappointing. I suspected thrombosis and held out no hope of recovery. She lingered for a week, and post-mortem I found a splendid specimen of thrombus in the usual position and teeming with larvæ.

Case IX. A case that I imagine supports the suggestion that the disease may be mistaken for ricked back was that of a four-months-old, beautiful, half-bred colt foal, that certainly showed the orthodox symptoms of back trouble, but was otherwise in good health. There was no history of an accident. I informed the owner that, although it undoubtedly looked like ricked back, it might eventually turn out to be a case of developing thrombosis. I applied a charge across the loins, not, however, with much hope of it improving matters. Meeting the owner a few months later he told me that the foal did no good, and about a month after my visit got colic, became paralysed, and died suddenly. I believe this was another instance of thrombosis, and that the symptoms of ricked back were the result of the gradual formation of a thrombus.

I could record several other cases with a somewhat similar history, but I regret I have never had an opportunity of making a post-mortem to confirm or refute my suspicions.

Case X. Another interesting example of the disease, and one I failed to diagnose at the time, was a big two-year-old half-bred colt. He had been gradually losing condition for some weeks and ate very little. I diagnosed indigestion, and prescribed for him accordingly. He never made any progress: gradually wasted away, became weak on his hind limbs, and died in a fortnight, after lying for a few days. The post-mortem presented a picture I had never seen anything like before. The cæcum was greatly enlarged, blue-black in colour and its wall was everywhere about two inches thick. There were also numerous large ulcers on the mucous surface. It seems strange to me now not to have suspected thrombosis at the time.

Case XI. A somewhat similar case was that of an old thoroughbred mare called Croquet, the dam of Jenkinstown, that won the Grand National. On three occasions, with about a fortnight's interval, I was called to attend her for colic, but each time she recovered before I arrived. She was in foal, was losing condition, and the appetite was suppressed. A few weeks before her time she gave birth to a dead foal, and died almost immediately afterwards. The stud groom sent me word and I went and made a post-mortem. A considerable portion of the bowel—what part exactly I didn't note—was black, very thickened and almost gangrenous. Again I was at fault.

Case XII. A very valuable brood mare, winner of several classic races, and valued by the owner at ten thousand pounds, was noticed uneasy at grass and was brought in and given a quart of linseed oil. As she did not improve I was sent for. It seemed to be a subacute case of colic, due to impaction. She did not improve with treatment and every possible attention. I stayed with her all night and she seemed somewhat better towards morning. About mid-day, however, she became very much worse, and I suspected twist. She died in great pain that evening. On post-mortem examination next morning there was no impaction or twist; there was some fluid in the abdominal cavity, containing small portions of ingesta, the result of a slight rupture of the double colon, which I then suspected as the cause of the trouble, although there were no signs of peritonitis. I believe now the rupture only occurred a few hours before death and that anæmia of the bowel predisposed to it. I did not think of examining the mesenteric artery, but in the light of subsequent events, I am convinced the mischief was there.

Cases XIII. and XIV. About five months later, on the same stud farm, I had two cases of the disease in two valuable thoroughbred foals, each six months old; three days separated the attacks. Both showed slight stiffness behind when walking. They lingered on for a few days, showing symptoms of dull pain.

One of the specimens on the table is the anterior mesenteric artery of the second foal; that of the first was also badly affected These two cases suggested to me that the mare died from the same cause, notwithstanding the difference in symptoms. While the last foal was ailing two companions of his were observed not to be feeding, and were taken in. For a few days they showed uneasiness at times by pawing; the appetite was capricious and the temperature 103°; yet all the functions were apparently working normally; both recovered. I consider these were two mild cases of the disease.

Case IV. The last case I shall record occurred two months ago, and differs considerably from all the others. It was a seven-months-old thoroughbred colt that was noticed walking very stiffly behind. When he lay down he could only rise again with great difficulty, but he showed absolutely no symptoms of pain, and seemed right in every other respect.

I diagnosed thrombosis and held out very little hope of recovery. He ate fairly well next day, was not uneasy, but continued to walk as if his back was injured, and had to be assisted to rise; when down he always lay prostrate on his side. The following morning the groom found the foal dead in the stable, lying just as he left him the night before; he had apparently died without a struggle. I travelled fourteen miles to the place at my own expense to make a post-mortem examination and think I was well repaid for my trouble. I have here his mesenteric artery, which, along with all its branches, seems to be completely closed by a thrombus, rich in larvæ.

I could instance numerous other cases, many of them presenting interesting phenomena, but those I have recorded will, perhaps, be sufficient to illustrate a mental picture of most of the many peculiar forms in which the disease may manifest itself.

Diagnosis. As I have already indicated, the history and symptoms of individual cases of this affection probably differ more widely than in any other disease; diagnosis is, consequently, often very difficult. It is supposed to be most commonly confounded with colic, and while this may possibly be true, I consider it may easily be mistaken for many other abnormal conditions. I must confess that at one time or another I have diagnosed twist, impaction, indigestion, kidney trouble, ricked back, and tetanus, where I now know for certain in most cases that a thrombus of the anterior mesenteric artery was the cause of death, and I have little doubt that in other cases the same disease had attacked the mesenteric or, possibly, some other important artery.

Where the attack is acute and all the ordinary causes of colic are seemingly absent, we should suspect the presence of thrombosis. Especially in the young, stiffness or partial loss of power behind or complete inability to rise should always suggest it. Repeated, short attacks of colic should also make us suspicious. Diagnosis by examination per rectum is, I consider, extremely difficult, if not impossible. In foals the narrow lumen of the bowel prevents the hand from being introduced far enough to palpate the artery properly. I ruptured the intestine on one occasion it attempting it. Some authorities state it is possible, but I have tried it many times without success. In older animals the artery is often out of one's reach. Perhaps, the fact of my arm being short accounts for my failure.

In all cases of sudden death, especially in young animals, where the cause is not obvious, the mesenteric arteries should be examined.

Treatment. From the nature of the disease treatment is of little use, and I am afraid it is never likely to be otherwise.

In mild cases, where it is possible for the circulation to be restored, it will not be necessary to interfere—provided we are confident of our diagnosis, for nature will do the needful without our assistance. Of course, it is almost impossible to distinguish such an attack from a mild case of colic.

This fact should make us examine our conscience as to whether it has always been those shot-gun colic draughts that have brought about such a wonderful and speedy recovery in many cases of apparent colic.

Prophylaxis.

Our only hope, therefore, lies in prevention, and in this we are up against a very stiff problem, especially if, as I have suggested, the disease is often disseminated through the medium of ordinary moist grass.

No precautions should be omitted. Animals ought not to be allowed to drink dirty water or to have access to stagnant pools. Horses in whose fæces the mature worms have been observed, or that are suspected of harbouring them in their intestines, should be separated from healthy stock and put under a course of treatment, notwithstanding the difficulty of getting rid of the mature parasites by drugs; all animals under suspicion should be isolated till they are considered free of the worms. Marshy pastures should also be avoided, suspicious fodder condemned, and the custom of removing droppings from the stable as soon as observed will remove a possible danger. Common sense hygiene should be practised and boxes and stalls periodically cleaned out and disinfected.

Discussion.

The Chairman: You have heard this very interesting and instructive paper by Mr. Magee: Usually such papers from him are very practical. It is a great

matter to have such facts and specimens laid before us. I may say that I never had any idea that there was so much of this disease. I am sure we will be very glad to hear any remarks that can be made on this valuable paper in which Mr. Magee has given us an account of his experiences and observations.

Prof. CRAIG: Mr. Chairman and Gentlemen—Mr. Magee has brought to our notice a very interesting lesion produced by one species of the most important family of worms (*Strongylidæ*) which are parasitic to equines. This family of parasites causes more damage in this country to the breeding and rearing of horses than any specific disease to which they are subject. With regard to the symptoms produced by the lesion which Mr. Magee describes, a good deal of difference of opinion exists. One has got in the first instance to remember that aneurism and thrombosis of the anterior mesenteric artery is very common in the horse. I have seen this lesion in 80 per cent. of horses in the dissecting room and at post-mortem examinations during the past twelve or thirteen years. We may therefore take it that in four out of every five horses which die from any cause whatsoever this condition will be discovered. It is therefore necessary to make a very careful examination before attributing death to this cause. In the large proportion of horses in which I found an aneurism of this artery and which a history was available, no symptoms were observed which could be traced to this lesion. At the same time I have no doubt that when the infection is a very heavy one and the thrombus is very large, it does interfere with the blood supply of the large bowel, and causes serious disturbance and symptoms of colic. Emboli from the thrombus may be cast off and produce even a fatal embolic enteritis. This does happen occasionally in young horses. But I do not think that this a common cause of colic. Colic appears to me to be more commonly induced by irregularities in the food, in dieting or work, and precautions taken with regard to these points, neglecting worms altogether, will reduce the number of such cases to a minimum.

Paralysis as a symptom of this condition requires some explanation. It does not seem reasonable to suppose that it is due directly to interference with the blood supply of the bowel, it may be accounted for by debility due to the action of the parasites in the bowel, or perhaps it may be caused by thrombosis of the termination of the aorta or its branches. This thrombus might be set up indirectly from the affection in the anterior mesenteric artery.

The chief symptoms caused by the Strongylidæ generally are those of anæmia, loss of condition and diarrhœa, and these may be attributed largely to the larval worms, in the bowel wall and to the hæmolysins which these worms produce.

In the cases of thrombosis of the anterior mesenteric artery which I have examined I have noted that the right division of that vessel was invariably involved. This is the division which supplies the cæcum and large colon in which the adult worms are found. This may indicate the route taken by the worms after ingestion by the horse. I have never seen the branches of the left division involved.

The worms present in the thrombus and aneurism are the larvæ and young adults of the *Strongylus vulgaris*. I do not know of any other species being found in this situation.

In dealing with the family of Nematodes to which Strongylus vulgaris belongs it was customary up to a few years ago to describe only two species, *Strongylus* (*Sclerostomum*) *armatus* and *Strongylus tetracanthus* (now called the *Cylicostomum tetracanthum*), but it has been shown by Loos and other investigators that there are at least 19 closely allied species of Strongylidæ in the horse. Of the armatus type there are three species, of which Strongylus vulgaris is one. No doubt that indicates considerable progress in our knowledge of these parasites, but it is not accompanied by a corresponding increase in our knowledge of their life history. We know that the adults of all these worms live in the lumen of the cæcum and large colon. There they lay eggs which are voided with the fæces. On the ground, in favourable conditions of moisture and temperature, embryos hatch out. I have seen the embryos develop in a few days in the fæces of affected horses when kept moist and protected from sunlight. They may be recognised in films of the fæces under a low power of the microscope. After a time the larvæ form and undergo at least two ecdyses. When they are first hatched out they are easily destroyed by desiccation, but later they become very resistant and withstand prolonged drying and considerable changes of temperature. It is probable that in this later stage the larvæ can live a long time on the ground, perhaps as long as two years. Not until the larvæ have undergone this development are they able to infest equines and pass through the remaining stages of their life history.

They are taken in with the food, particularly grass, and in water. A point of importance to note in the study of worms is that each worm in the body represents one larva taken up. The worms do not reproduce indefinitely in the body of the host. The degree of infection of the horse therefore depends upon the number of larvæ ingested with the food or drinking water.

The great majority of horses are infected to a certain extent with the Strongylidæ, but it is only when the infection is excessive that serious trouble is set up. This is thus associated at pasture with the degree of contamination of the ground with fæces teeming with the larvæ, and in that way is often due to over stocking.

What route the larvæ taken after being ingested by the horse is at the present time only a matter of conjecture. Judging from the development attained by the Strongylus vulgaris in the anterior mesenteric artery, which would prevent their passage into the bowel, one naturally concludes that these worms have strayed from their usual route through the body, *i.e.*, have been sidetracked.

As to the treatment of the disease caused by these worms, it may be said that there is no agent which will act directly on the parasites in the anterior mesenteric artery or in the bowel wall. Atoxyl has recently been well spoken of in the treatment of these cases. It may be given in 7 to 15 grain (½ to 1 gramme) doses in 1 to 3 per cent. solution hypodermically for a few days. It deserves a trial in this country. Arsenic is also used, but it seems to me that to be effective it should be used in larger does than are at present in vogue. Five grains is a common dose recommended, but there can be no doubt that a horse will stand 10 to 15 grains every other day unless it be in the last stages of the disease; 15 to 30 grain doses are used commonly in India in the treatment of surra. Tonics containing iron compounds and a nourishing diet are specially recommended to combat the anæmia and loss of condition resulting from a severe attack of these worms.

No great advance of recent years has been made in the direction of preventive measures. It is necessary to avoid over-stocking. If the owner is compelled to use infected fields for grazing purposes the horses should be allowed free access to rock salt, and should be given period doses of anthelmintics. Attention must be paid to the drinking water to avoid contamination.

Where serious losses from this cause have occurred, all the equines should be removed from the fields and the latter left vacant of equines for a period of two years, to allow the larvæ to die out. Ruminants, since they do not act as hosts of these worms, might be put on

the land in the meantime. To ensure the extermination of the larvæ, lime or salt in the proportion of 5 cwt. per acre should be applied over the fields. This is especially necessary where the owner desires to use the fields for horses at an early date.

Mr. HOWARD: I have first to compliment Mr. Magee on his choice of what is possibly the most interesting subject that has come before the Association for a great number of years. He has touched a subject of national importance. We know that this country prides itself on being a horse producing country, and this particular trouble possibly causes more loss to horse breeders in Ireland than any other trouble in horses. Prof. Craig has told you how it is to be dealt with, and settled the question of the particular worm. Like Prof. Craig, I am sorry I cannot agree entirely with the essayist in believing that these worms are responsible for all the troubles that Mr. Magee put down in the bill. I hope he will not be vexed by my saying that he assumes too much. We have heard veterinary surgeons and amateurs in cases of lameness talking of rheumatism. Well, "rheumatism" it appears covers a multitude of sins in the way of lameness. Mr. Magee may be taking to himself the happy relief of blaming this little worm for all the cases of colic he could not explain otherwise.

I could understand thoroughbred mares being affected on lands very badly poisoned with this disease—farms that have become "horse sick." The ordinary countryman understands that. I do not think that the disease is common in the older animals. I can quite see how a mare living on a place all her life may become troubled by the disease, but it seems that its ravages are more confined to young animals.

Mr. MAGEE: I would like to hear Mr. Howard explain the paralysis in these two aged animals I referred to, which were found lying in the stable and unable to get up, and yet they were perfectly right the night before. No form of colic that I know could explain that.

The CHAIRMAN: That is just what I wanted to know.

Mr. HOWARD: Any man that owns horses, or has had anything to do with them, knows that, even if you had them in a glass case with a man minding them, they will get into serious difficulties that cannot be explained. If I had an animal over five or six years old in good condition, and found him down to-morrow, unless I found out the actual condition, I would be very slow to believe that the worms had anything to say to it. I believe that if the worm is to be blamed for it the animal will be showing symptoms for a considerable time before to the careful observer. I am satisfied of that, but the fact of my being satisfied will not satisfy Mr. Magee. There is no doubt whatever that farms where the owner has gone in extensively for producing young stock in large numbers in the year the land may become poisoned, or "horse sick." That has been recognised for some years now. Hence the superiority of horses reared in Ireland has been simply because you have had few horses on the farm, and these were brought to top mark. On the so-called stud farms as recently established in Ireland, where a man has attempted to rear a large number of horses on a limited area, you will have this trouble. One possible way it gets in is through mares that have become already affected with the disease, being brought it. In that way, and in no other, could be explained what Mr. Magee found in the case of a six months old foal. That animal was well fed and in good condition.

There is the disease which in the West of Ireland is called "water farcy." Colts and foals there suffer from this disease in the winter time, and it is attributed entirely to the presence of these worms in the bowel. The belly drops down and you can sink your finger an inch into it, and the impression will remain. It is marvellous the number that recover, and develop into good animals and live to a good age. It is likely that where severe thrombosis sets up in some of the arteries the animals will not recover, and very often they die without developing paralysis. Mr. Magee has described other cases where they have been for several days unable to get up. I believe the ravages of this worm or family of worms is to be accounted for on good land and good farms by their being simply poisoned.

Mr. MAGEE: Being overstocked?

Mr. HOWARD: By "horse sickness." A good many 'cute men here in Ireland will tell you that they never reared a good horse where they were overstocked. On the best managed stud farms they always make it a point to have a certain number of cattle and only a few horses running with them, so that the farm will not become "horse sick."

I cannot agree with Mr. Magee in assuming that the case of back trouble would be due to the presence of thrombus. It is very hard to explain how thombus in the mesenteric artery would even block up the whole blood supply and cause the back symptoms. Any man mixed up in the rearing of young horses knows how prone they are to have a tumble and be "wobbly" next day, especially in the case of a valuable one. You cannot advise a horse how to conduct itself. If you find a horse doing well to-day and he is "wobbly" to-morrow, it is very hard to blame worms. The horse may have done something foolish. Local symptoms would be more likely if thrombus were the cause.

The CHAIRMAN: I do not quite understand the cause of the paralysis.

Mr. MAGEE: Where paralysis accompanies the colic it is uniformly fatal.

Mr. HOWARD: I was not discussing that at the moment. Mr. Magee, in his paper, was assuming that in cases of "jink back" the symptoms were due to thrombus. I believe that he actually found thrombus.

Mr. MAGEE: No, unfortunately, I did not.

Mr. HOWARD: Therefore, I think it is all against the assumption. I do not think it fair to assume from the back symptoms that thrombus was present. We all know how prone young animals are to tumble in the field. Unfortunately, I have had more than one of my own do that sort of thing and hurt their backs. There is not a year passes that I do not see a great many.

The CHAIRMAN: They get better, don't they?

Mr. HOWARD: Only one per cent. become of use. The other 99 per cent. are better shot.

Mr. MCCANN: They do not die, like Mr. Magee says!

Mr. HOWARD: They don't die.

Mr. MAGEE: Can you recollect any that did die?

Mr. HOWARD: Oh, I can.

Mr. MAGEE: If you can, it is evidence in favour of my assumption. Can you recollect any cases of "jink back" which died within a reasonable time?

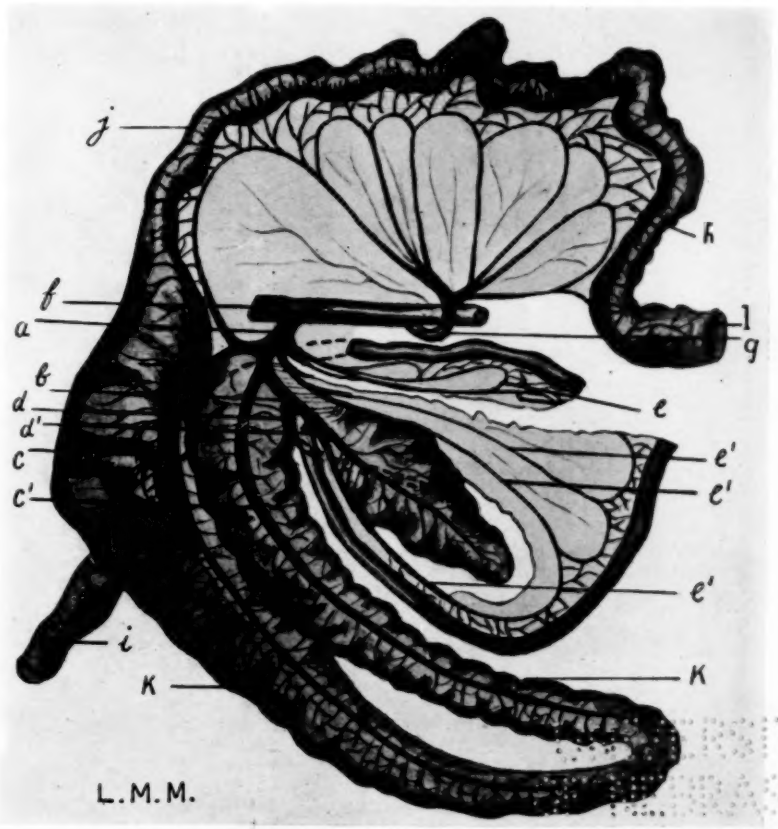
Mr. HOWARD: I cannot recollect. Yesterday, I was re-called to a case of "jink back" I had seen six months ago. I am arguing against your assumption. I do not think it is fair without having found the presence of these things to assume because an animal is in perfect health to-day, and you find him "wobbly" to-morrow, that it is due to the presence of thrombus. Or, even with the presence of thrombus, that he did not tumble the wrong way.

Regarding those that die, last April I castrated five, and amongst them was a nice chestnut colt. Just as he walked out of his box I did not like the way he was putting his hind legs. I said to the groom there was something wrong with the colt's back. "I am very glad I have seen it, because in a fortnight's time we might be blaming ourselves if it was not discovered now." He went down surely enough, and I saw him yesterday, and I told the groom he might as well have him shot. He was in good condition, but what the horsey fraternity call "a bobby."

1 / 1st North Midland Divisional Mobile Veterinary Section.

MESENTERIC ARTERIES OF THE HORSE.

(*a*) Anterior mesenteric artery.
(*b*) Ileo-cæcal artery.
(*c*) Dorsal colic artery.
(*c'*) Ventral colic artery.
(*dd'*) Arteries to cæcum.
(*e*) Jejunal arteries.
(*e'e'*) Branches to ileum.
(*f*) Posterior aorta.
(*g*) Posterior mesenteric artery.
(*h*) Rectum.
(*i*) Duodenum.
(*j*) Floating colon.
(*k*) Double colon.
(*l*) Anus.



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Prof. O'CONNOR: What do you think was wrong with the back in that case?

Mr. MAGEE: What do you consider a common or garden case of jink back is?

Mr. HOWARD: It is due I believe to an injury to the spinal column—probably one of the small bones.

Prof. O'CONNOR: It is a mystery to me what it is.

Mr. HOWARD: It is due to pressure on the spine. It is caused by being directly injured. I would not say the extent of the injury further than that it was one of the small bones, and that there was a certain amount of pressure owing to a deposit in that part.

Prof. O'CONNOR: That is merely an assumption of yours.

Mr. HOWARD: As regards treatment which we are fond of advising when we are called to see a case of "water farcy"—the animals are put on the most nourishing diet, with plenty of salt and iron. When they do fairly well they recover. I have known several horses to turn out good horses in that way although the abdomen was dropsical.

Mr. WILKINSON: I suppose these animals swelled in the legs?

Mr. HOWARD: Sometimes you will have the belly eight inches down and no swelling in the legs. I have seen the abdomen, and you could puncture it and a stream of serum flows out. I have a case in hand of a colt with dropsy. Whether he rubbed himself against anything I do not know but he has taken a patch of skin out of his abdomen in the centre eight or nine inches in circumference. I believe he is going to get better.

Mr. PRENTICE: Do you attribute that to what is called water farcy?

Mr. HOWARD: Yes. This condition may have been brought on in the first few days by hand-rubbing. There was no application of embrocation.

Mr. MAGEE: Can you give us some idea of what water farcy is?

Mr. HOWARD: I believe it is due to obstruction of the circulation owing to the presence of these worms in the bowel, and possibly in some of the arteries. I think the greatest possible thanks of this Association and the profession are due to Mr. Magee for the way he has followed up this particular subject. I do hope that we will be able to point to him as the man who has drawn some definite conclusions that will be accepted for the guidance of the profession in the future in reference to this disease. (Applause).

Prof. O'CONNOR: Mr. Chairman and gentlemen, I am sorry I cannot throw any enlightenment on this disease. I am sure we are all anxious to express our thanks to Mr. Magee for bringing forward this most interesting paper on such a very important subject. I particularly owe him thanks as he has got me out of a difficulty. Mr. Winter had promised to read a paper but since then he had arranged to go to Canada, consequently I wired to Mr. Magee, and he replied at once that he would provide a paper. I had an idea he would give us some information on this subject, as he had been telling me about the number of cases he had.

Just a short time ago I was called in consultation about some cases similar to these. Several animals had already died and others were very bad. I saw some of them that were bad, and the striking symptoms about them were that they were very weak, anæmic and emaciated. Most of them had a watery diarrhœa. The mucous membranes were pallid. Some were unable to rise without assistance.

The CHAIRMAN: Was there any œdema?

Prof. O'CONNOR: There was in some of the cases. The owner told us that he had had horses on the farm where these animals were for years and he never had a case like these before. This was the first year that a large number of young animals were put on it. The colts were uncastrated.

Mr. HOWARD: It would be interesting to know what happens for another year or two.

Prof. O'CONNOR: They were bought in different parts of the country and commenced to show symptoms soon after being put on this farm. The owner's veterinary surgeon suggested that there were too many animals on the farm, and that they had not enough grass: and he prescribed generous hand feeding and warm and tonic medicine. Some cases improved, but others got gradually worse and died.

With regard to jink back, the worst of Mr. Howard's argument is that he has nothing to go on against Mr. Magee. They are both going on assumptions. I would be very glad to get some explanation of these cases of "jink back"—or suspected cases—which do not get better. The fact that they do not improve would seem to point to some unusual cause, for, if they were the result of comparatively slight injury, one would expect the animal to get better.

Mr. HOWARD: Have you actually seen cases get better that had really an injured back?

Mr. WILKINSON: You mean real "jink back"?

Mr. HOWARD: I mean the real thing, where the horse was seen to hurt himself.

Prof. O'CONNOR: If you have proof that the horse hurt himself and that he does not get better, you have evidence of the cause; but if you have to take the owner's history of a case you cannot place great reliance upon it.

Mr. HOWARD: Have these cases of jink back been examined?

Prof. O'CONNOR: That is what I am wondering about.

Mr. MAGEE: I examined one. It was the case of an aged horse which had a fall in hunting. I examined the spine and psoas muscles and found chronic strain.

Mr. EBBITT: I was much interested in Mr. Magee's paper. We have in America a disease known as pernicious anaemia or swamp fever. Those of us who were in the Philippine Islands and had an experience with Surra thought that a Trypanosome would be found in the blood of animals affected with swamp fever.

We sometimes found young animals down in the pasture unable to rise, and Dr. Peters, of the Nebraska experiment station, found the sclerostoma tetracanthum in the posterior aorta. For a time, at least, it was thought to be the cause of swamp fever. There may be some confusion of one disease with the other. As regards Mr. Howard using the word farcy, or water-farcy, I think that name should be eliminated from the vocabulary of the Veterinary Surgeon, as it always has a tendency to be confounded with glanders.

Mr. MCCANN: Only a couple of years ago I castrated a number of colts, one of them had a staring coat and was not so healthy-looking as he should be. I remarked it to the man in charge at the time I castrated him, and after three weeks the owner told me that the colt was very stiff and drowsy-looking; and he asked me if the castration would have anything to do with it. I had told him that there might be a little swelling, and to be sure that he got exercise. I asked him if there was any swelling, but apparently the colt got over the castration all right. I informed the owner that I considered the trouble was probably due to the presence of worms in an artery, and I said, "If you like, treat him with worm medicine." The colt got stiffer, especially behind. He grew gradually worse, became paralysed and died. I did not make a post-mortem because I was very busy at the time: but I feel confident that thrombosis was the cause of the condition. In connection with "jink back" I recall a case in an old mare. I examined the psoas muscles, and there was distinct

tenderness. She never recovered from the "jink back," but is able to work on the farm. I have nothing further to say except to thank Mr. Magee for his very interesting paper.

Mr. WILKINSON: I am afraid I have not paid sufficient attention to this subject. Many a time I thought I had a case of inflammation of the bowels to contend with, and after post-mortem examination I have felt rather small, as often the symptoms which I expected to find have been absent. Possibly aneurism was present in some cases, and in future I will look more carefully for it. I cannot quite follow the argument that this condition would give rise to paralysis of the hind quarters. I get a great many colic cases to attend, but not many in young horses. At times I have had very considerable difficulty in satisfying myself as to what the cause was, and possibly I have overlooked the condition of which you speak.

Mr. MACLEAN: Two and a half years ago I was called in to see a yearling colt which I had castrated a month previously. On arrival I found the colt down in a field and unable to rise. The owner told me that a couple of hours previously he noticed the colt galloping in a circle. He was apparently in normal health. Suddenly he dropped and did not rise. He suspected tetanus. On examination I found no trace of tetanus. We tried to get the colt on to his feet, but he could give us no assistance. Treatment was of no avail. Fæces and urine were retained. I diagnosed thrombosis and advised destruction. The colt was put out of pain, and I proceeded with the post-mortem, confident that my diagnosis would be borne out. Alas! I found no thrombus in the mesenteric, just three or four sclerostomes, and no signs of thrombosis of any of the branches of the anterior mesenteric artery. In the walls of the bowel were calcareous nodules, but not in numbers sufficient to cause trouble. Needless to say, I did not impress my client very favourably; and I am still at a loss to know the cause of the trouble.

Mr. WATSON: I would like to associate myself with the previous speakers in congratulating Mr. Magee on his very able paper. I agree with Mr. Howard in saying that we have not had a more interesting paper for a number of years. Like Mr. Wilkinson, when I was in practice I was often at a serious loss in making post-mortems to satisfy myself as to the cause of death. One fact has impressed me for a number of years, and that is, in reading the medical evidence at a Coroner's inquest, how quickly they arrive at the cause of death. How easy it is to say "fatty heart." It puts me in mind of a butcher they had in O'Keeffe's, the knacker's yard, for a number of years. Whenever a veterinary surgeon went there to hold a post-mortem examination the knackers' butcher always had his opinion ready as to the cause of the death of the animals brought there. He would say without hesitation, "Here you are, Sir, rupture of the diaphragm," or "Here you are, Sir, twist of the bowel." I remember going to make a post-mortem with the late Mr. Mat. Murphy, sen., who said "Now John, no rupture of the diaphragm or twist of the bowel, we are really going to find out the cause of death." What really impressed me in connection with Mr. Magee's paper is that it is only men like Prof. Craig who can follow up the smallest capillaries in the body who are competent to make an exhaustive post-mortem examination. Mr. Magee has given us food for reflection and as Mr. Wilkinson said, in making post-mortems we should examine and follow up all the arteries to see what really is the cause of death.

The CHAIRMAN: I can only congratulate Mr. Magee on his excellent, most interesting and original paper. There seems to be some little difference of opinion between Mr. Howard and Mr. Magee. The former does not consider that the paralysis which appeared in the later stages of the cases referred to was the result of the thrombosis which was found on post-mortem. The theory advanced by Mr. Magee, whether right or wrong, seems to me to be a rational one. The thrombus obstructing the flow of blood to a portion of the intestine would render the part anæmic and perhaps ultimately gangrenous, and these conditions would allow or facilitate the passage of toxins and micro-organisms into the peritoneal cavity, and thence by absorption to the spinal cord, or nerves in that region, and thus cause the paralysis. Whether Mr. Magee is right in inferring that the many similarly affected cases which he met with, and in which no post-mortem was made, were cases of thrombosis is another question. In the district in which I happened to practice the land generally was well cultivated, consequently, I met with few cases of thrombosis or aneurism. I have from time to time, extending over a lengthened period met with an occasional case of paralysis, the nature of which was rather mysterious. The initial symptoms were those of ordinary colic. In the course of a very few hours the horse would show signs of weakness in the hind quarters A little later and he would only be able to prop himself up on his fore limbs, and soon after he would be found lying flat on his side, perspiring, dashing his head about and often tearing up the ground with his fore feet. Such cases usually terminate fatally in from 15 to 48 hours. At the onset the symptoms are by no means violent. Sometimes one gets as much credit by giving an early and correct opinion as to the termination of the disease as if he had the good fortune to succeed in effecting a cure.

With regard to tetanus, in its earlier stage when there was only a suspicion of stiffness I have always found an unexpected tap under the horse's chin a reliable test for the disease. The protrusion of the membrana nictitans always accompanies the slight tap.

I have never found "chink" back result from casting a horse. I have always been careful to see that the hind pasterns were pulled to, or braced in, a position near, but not too close to the girth There is, I think, always some risk when the horse is allowed to extend his hind feet too far forward.

REPLY.

Mr. MAGEE: I am more than pleased with the discussion which the paper has produced and with the way my little effort has been received. Prof. Craig discussed the disease in all its phases, and we are greatly obliged to him for the information he has given us, especially on the scientific side of the subject I am also glad that he agrees with me as to the frequent occurrence of the disease, and its seriousness. Perhaps the most discussed point to-night—and very few agree with me on it—is that the disease may cause either slight or extreme paralysis. Nothing that has been said, however, has shaken my confidence in what I must for the present call this theory.

The CHAIRMAN: We do not doubt it; we cannot, perhaps, understand it.

Mr. WATSON: Say "not proven."

Mr. MACCANN: It was proved by the essayist in a good many cases.

Mr. MAGEE: I am confident that thrombosis of the anterior mesenteric artery causes paralysis in a lesser or greater degree. Naturally, you will say to me, "How do you explain it!" I admit it is somewhat difficult to do so, but I don't think it is so difficult to explain in an acute case. There is a paragraph in the paper which I have read that touches on the point and, to my mind, does give an explanation of it. It says, "The intestinal wall which is damaged in its nutrition, or eventually becomes necrotic through grave circulatory disturbances, early permits intestinal bacteria to enter into the peritoneal cavity; these then multiply rapidly in a serous exudate and produce circumscribed, and later, general

peritonitis, which causes continuous inflammatory pain; the bacteria and their toxins are also absorbed into the blood, and general infection or intoxication with their symptoms follow." That contains an explanation of it. A large portion of the intestine becomes affected when its blood supply is cut off; no fresh blood is going to the part, and so the bacteria in the bowel multiply without hindrance and produce their toxins; they easily pass through the necrosed wall of the bowel, and are then carried through the system by means of the veins, or, possibly, the lymphatics, their secretion eventually causing intoxication and paralysis. In acute cases it is simply an intoxication.

The CHAIRMAN: Affecting the spinal cord?

Mr. MAGEE: Affecting the nervous system. While thinking about this subject lately, the idea occurred to me that possibly there might be some connection between this intoxication caused by a thrombus and the intoxication which we call milk fever in the cow—a disease which we can cure, although we don't understand its exact nature. It would appear to me that both are brought about by interference with the circulation. It is certainly so in thrombosis, and possibly it may also be the case in milk fever. One of the first symptoms often observed in the latter disease is a partial, sometimes almost a complete suppression of the milk supply. Now the udder is simply a large gland, and when the blood supply to a gland is interfered with its secretion must necessarily suffer. Therefore, I think it is probable that owing to some influence on the blood vessels of the udder a condition of congestion is brought about which causes anæmia of the gland or part of it, resulting in the formation of toxins which bring about the intoxication and paralysis. How our present method of treatment restores the blood supply of the gland to the normal again I admit requires a lot of thinking out. I believe, however, that there is something in this suggestion, and that it is worth considering.

Prof. CRAIG: In these cases did you see any change in the bowel wall?

Mr. MAGEE: Yes, in several. In that case of the two-year-old, where I diagnosed indigestion, the cæcum was absolutely black, enormously thickened, with ulcers scattered over the mucous surface. The colt was paralysed for a few days before he died.

The next point is that discussed by Mr. Howard regarding "ricked" or "jinked" back, or the horse that is called a "bobby." I suggested that a case of developing thrombosis may be easily mistaken for "ricked" back. Mr. Howard asks me how would I explain that the thrombosis could cause this condition which resembles "ricked" back. This slight paralysis is, perhaps, more difficult to account for than the complete paralysis we meet with in acute cases of the disease. Supposing a thrombus completely destroys the function of an artery such as (*c*) which nourishes a large portion of the bowel, and circulation is destroyed, even only temporarily. Although the blood supply is restored eventually by collateral circulation (*c'*) the thrombus remains permanent and the bowel cannot be receiving its full complement of blood. I think that it is natural to conclude that the affected intestine is not as vascular or in as healthy a condition as before, and that its function is impaired. Consequently, a certain amount of toxin is being constantly manufactured and absorbed, causing a mild auto-intoxication which produces this weakness or partial paralysis behind.

Mr. HOWARD: Why not in front, as in milk fever?

Mr MAGEE: At present I admit I cannot satisfactorily answer that.

Another point I would like to mention is that a portion of a horse's bowel being in this condition would, I consider, predispose him to frequent attacks of colic, as its normal secretion, absorption and peristaltic movements would be interfered with; it might also prevent him putting on condition. Mr. Howard said I was assuming a great deal and that in many of the cases I recorded, quite possibly I was not correct as to the cause of death. I quite agree with him that I have assumed much in those cases in which I was unable to make a post-mortem examination, but in each instance where I was able to make a post-mortem I feel certain that when I found thrombosis of the anterior mesenteric artery it was the cause of the animal's death.

It is a great thing at a post-mortem to find something you can "hang your hat on." When you can show your client the enormous thrombus in the artery, completely blocking it, and the little worms scattered through the clot, it is very satisfactory.

Prof. Craig spoke of the way in which the larvæ are thought to make their way into the anterior mesenteric artery and of what becomes of the worms ultimately. If the theory be right—that these are larvæ that have gone astray and take no further part in the continuation of the species, I think it would be hard to reconcile it with the case described by Poppel, where a ten days old foal had a thrombus as large as pigeon's egg. I have read of another theory which suggests that infection is possible from the uterus. It seems, perhaps, far-fetched, but really not more so than our views on other diseases: in gid in sheep, for instance, as to how the parasite reaches the brain. The suggestion is that the worms pass from the mother to the fœtus in the blood.

The CHAIRMAN: You do not mean directly from the exterior.

Mr. MAGEE: From the mother to the fœtus while in utero.

I remember once when Prof. Hobday was operating on some roarers at Strokestown, in Roscommon, Mr. Ryan showed us a colt apparently suffering from ricked back. He said there was no history of an injury. Prof. Hobday said it certainly looked like ricked back, and when I suggested that possibly it might be a case of developing thrombosis of the mesenteric artery, and told him of other suspicious cases I had had, he became very interested. I met Mr. Ryan at the following horse show in August and asked him what happened to the colt. He said that about a month afterwards the colt suddenly got colic and died. Of course, that is by no means confirmatory, but in view of many other cases with a similar history it looks, at least, very suspicious.

Recently I was consulted about a thoroughbred colt foal, five months old, that was apparently suffering from ricked back. I told the owner that possibly it was a case of thrombosis, and explained the nature of the disease. As the foal was insured, and he was anxious to have him destroyed if the case was hopeless, he suggested a consultation. The Consultant was a man much older than myself, and the moment he saw the foal, he said it had ricked back and should be shot. I suggested to him that it might be thrombosis, but he simply held his tongue and looked wise; I don't believe he ever heard of the disease. The Company would not agree to the destruction of the foal till their veterinary surgeon saw the animal, and some days later he and I attended, and he considered it a case of ricked back, and advised slaughter. I, there and then, shot the foal with the humane killer, and proposed we should make a post-mortem examination; as I said, I had the courage of my convictions. The veterinary surgeon for the insurance company, however, said that he thought it would not be fair to do so in the absence of the consultant veterinary surgeon. I, therefore, did not press the point, and there was no post-mortem. You will say, perhaps, there was nothing there to suggest thrombosis. Well, there is something else that made it very suspicious. When the consultant veterinary surgeon and I, on the first occasion, had seen the foal, the owner

drew our attention to a four-year-old, half-bred colt that was also showing signs of weakness behind. I said that possibly the two cases were similar; the consultant said he didn't think it of much importance, and advised turning him out and forgetting about him. He was turned out, but they couldn't forget about him, as he gradually wasted away, became paralysed and died. I regret to say the owner did not tell me about him till after his death. I think it is fair to assume that these were both suspicious cases.

I have not the slightest doubt, either, that the cause of death among the young horses that Prof. O'Connor told us about was this disease.

Mr. Ebbett found some fault with Mr Howard's nomenclature of diseases. I am afraid we cannot always avoid calling diseases by their common names. If we call them by their technical names our clients will simply think we are talking through our hats. There is nothing that the layman objects to more than using jaw-breaking names that he does not understand.

Mr. Ebbett referred also to thrombosis of the iliac arteries. I have never had a case of that. I think the explanation of the temporary paralysis, which is a symptom of the condition, is that temporary anæmia is caused, and that anastomosis restores the circulation again; possibly, also, the blood may pass slowly through a small tunnel in the middle of the thrombus.

Mr. Dunlop said he never had any difficulty in diagnosing tetanus. Well, I really think there are very few diseases that are not difficult to diagnose at some time or other. That is my experience. For instance, you will sometimes find a case of tetanus showing no typical symptoms; just a stiffness of the hind limbs, where, if you had not had previous experience of such a case you would not think of applying the well-know test of suddenly raising the head to see if the membrana nictitans would shoot across.

The CHAIRMAN: Even with the mildest case I have never known that test to fail.

Mr. MAGEE: Yes, but the thing is first to be able to suspect it in a very mild case, before other symptoms have developed. In conclusion, I have only to say again how pleased I am with the discussion the paper has provoked, and with the way you have received it.

Mr. WATSON proposed that the best thanks of the meeting be given to the Chairman for the manner in which he conducted the debate and for the valuable suggestions he had made on the paper.

Mr. HOWARD seconded the vote of thanks, which was passed unanimously.

Prof. CRAIG proposed a vote of thanks to the essayist (Mr. Magee) for his valuable paper, and the interesting specimens he had brought before the Association in connection with it.

Prof. O'CONNOR seconded the motion, which was passed.

A Substitute for Antiformin.

A perfectly efficient substitute for Antifomin, hitherto supplied from Germany, may be made by passing chlorine into 15 per cent. solution of sodium hydroxide to nearly saturation. The chlorine may be prepared by adding pure hydrochloric acid to manganese dioxide.

To make 1 litre, 1 lb. of pure acid (sp. gr. 1·16) and about 6 oz. of manganese dioxide are required. The generation of the chlorine is assisted by a gentle heat. The gas is led into the soda solution, which has been placed in a tall bottle, the delivering tube reaching to the bottom. Enough chlorine has been added when the soda solution acquires the colour of lemonade.

The cost per litre works out at about 1/1.—*Lancet.*

Slaughter of in-calf Cows.

At a recent meeting of the directors of the Scottish Chamber of Agriculture, Mr. G. A. Ferguson called attention to the fact that in one of the Glasgow abattoirs 60 per cent. of the cows slaughtered were found to be in calf; Mr. Ferguson was asked for details, to be communicated to the Board of Agriculture.

Promoted to be a non-com. five minutes after joining is a record just established by a Nottingham recruit.

No sooner had J. W. Smith, of Lenton Abbey, Notts, kissed the book in being attested a private in the Army Veterinary Corps than he was given the rank of quartermaster-sergeant. He held high credentials for the handling of horses and the doctoring of their ailments.—*Weekly News.*

DISEASES OF ANIMALS ACTS 1894 TO 1914 SUMMARY OF RETURNS.

Period.		Anthrax		Foot-and-Mouth Disease.		Glanders.†		Parasitic Mange. ‡		Sheep Scab.	Swine Fever.	
		Out-breaks (a)	Animals.	Out-breaks (a	Animals.	Out-breaks (b)	Animals.	Out-oreaks (b)	Animals.	(b)	Out-breaks (a)	Slaughtered. *
IRELAND. Week ended	Feb. 13	...	...	...	...	...	...	Outbreaks 1		16	6	69
Corresponding Week in	1914 ...	...	...	...	...	...	...	2		32	7	60
	1913 ...	...	...	...	...	...	...	4		21	6	5
	1912 ...	...	...	...	...	...	...	4		19	1	6
Total for 7 weeks, 1915	...	...	...	...	...	...	...	8		108	26	183
Corresponding period in	1914 ...	...	...	2	28	...	...	23		180	27	198
	1913 ...	...	...	...	...	...	...	61		135	34	167
	1912 ...	1	1	...	...	...	...	17		143	16	170

Department of Agriculture and Technical Instruction for Ireland, (Veterinary Branch), Dublin, Feb. 15, 1915

NOTE.—The figures for the Current Year are approximate only.

* As Diseased or Exposed to Infection

ARMY VETERINARY SERVICE

Extract from *London Gazette*,

WAR OFFICE, WHITEHALL, Feb. 18

His Majesty the King has been graciously pleased to approve of the undermentioned rewards for services rendered in connection with Operations in the Field. Dated Feb. 18, 1915.

To be Companion of the Order of the Bath.

Col. (temporary Brig.-Gen.) J. Moore, F.R.C.V.S.

* * * *

ARMY VETERINARY CORPS.

D.S.O.—Lieut.-Col. W. D. Smith.

Military Cross.—Sgt.-Maj. 88 W. Beckett and 103 F. A. Nason.

* * * *

In the Dispatch from Sir J. French, dated Jan. 14, the following names occur among those recommended for gallant and distinguished service in the Field :—

Major G. Conder.
Major E. E. Martin, F.R.C.V.S.
Captain F. C. O'Rorke, F.R.C.V.S.
Major F. W. Wilson, F.R.C.V.S.
Lieut. S. W. Marriott, Army Vety. Department (attached 34th Brigade).

Feb. 20.

REGULAR FORCES. ARMY VETERINARY CORPS.

T. Gordon to be temporary Lieut. Dated Feb. 1.
C. McPhail to be temporary Qmr., with the hon. rank of Lieut. Dated Feb. 21.

Feb. 24.

TERRITORIAL FORCE. ARMY VETERINARY CORPS.

C. E. W. Bryan to be Lieut. Dated Feb. 11.

OBITUARY

J. J. SPERRING, F.R.C.V.S., Birmingham.
Graduated, Lond : Dec., 1862 ; Fellow, July, 1882.
Mr. Sperring died on Feb. 20th. Aged 63.

JOHN MCDONALD, M.R.C.V.S., Banff.
Graduated, Edin : May, 1894.
Mr. McDonald died on Feb. 19th. Aged 42.

SOUTH DURHAM & NORTH YORKSHIRE V.M.A.

Dear Sir,

I much regret having omitted to mention in the report of the special meeting of this Association, which appeared in your issue of the 13th inst., that another of our members, Mr. P. B. Riley, of Barnard Castle, was serving in the A.V.C.

Mr. Riley, was on the reserve of officers, and on being called up for duty at the commencement of the war, at once left his practice, and is, I believe, at present in France.

Perhaps you would kindly publish this letter in your next issue, when possibly he may see that the omission has been rectified, and he will, I know, understand that the non-mention of him was quite unintentional, and I hope will accept this apology from me.

Thanking you in anticipation, Yours truly,

JAMES H. TAYLOR, Hon. Sec.

DISEASES OF ANIMALS ACTS 1894 TO 1914, SUMMARY OF RETURNS.

Period.		Anthrax		Foot-and-Mouth Disease.		Glanders.†		Parasitic Mange. ‡		Sheep Scab.	Swine Fever.	
		Out-breaks (a)	Animals.	Out-breaks (a	Animals.	Out-breaks (b)	Animals.	Out-breaks (b)	Animals.	(b)	Out-breaks (a)	Slaughtered. *
Gr. BRITAIN. Week ended Feb. 20		17	18			1	1			6	62	175
Corresponding week in	1914 ...	12	12	1	4	3	4	64	102	5	63	487
	1913 ...	15	16			1	1	90	165	10	29	280
	1912 ...	29	32			6	9	102	229	6	56	940
Total for 8 weeks, 1915		141	158			6	9			102	625	2596
Corresponding period in	1914 ...	149	162	1	4	18	42	679	1298	102	423	3786
	1913 ...	103	116			27	85	689	1504	89	267	3136
	1912 ...	206	234			23	46	1110	2712	114	483	6311

‡ The Parasitic Mange Order of 1911 has been suspended as from 6th August, 1914.

(a) Confirmed. (b) Reported by Local Authorities. † Counties affected, animals attacked : Middlesex 1.
Board of Agriculture and Fisheries, Feb. 23, 1915

IRELAND. Week ended Feb. 20	...	...	...	...	...	...	Outbreaks ...	13	7	16
Corresponding Week in	1914 ...	...	...	...	...	...	...	...	20	2	21
	1913 ...	...	...	...	...	...	...	2	26	...	4
	1912 ...	...	...	...	...	...	...	3	15	5	22
Total for 8 weeks, 1915 ...	...	...	...	...	...	...	...	8	121	33	199
Corresponding period in	1914 ...	...	...	2	28	...	...	23	200	29	209
	1913 ...	1	1	...	...	...	...	63	161	34	161
	1912 ...	1	1	...	...	...	...	20	158	21	192

Department of Agriculture and Technical Instruction for Ireland, (Veterinary Branch), Dublin, Feb. 22, 1915
NOTE.—The figures for the Current Year are approximate only. * As Diseased or Exposed to Infection

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CIVIL VETERINARY SERVICE.

The shortcomings of a hastily collected, untrained service, for duties requiring a special military training of at least a year, have, from time to time, been dealt with in the pages of this history. We have shown that to turn adrift in the Field some hundreds of civilians imbued with patriotism does not constitute an efficient veterinary service.

With few exceptions, the C.V. Surgeons were all young men, the majority having recently qualified. They were engaged for a year (p. 27d), at the end of which time they were free to re-engage or return home. Those who joined at the beginning of the war received the pay and allowances of a Veterinary Lieutenant, and two months' pay as gratuity on completing the year of service. Towards the end of the year 1900 it was evident this rate of remuneration was insufficient to retain the services of those now experienced. In June, 1901, it was decided that better terms must be offered; an increase in pay of 5/- a day was given after eighteen months' service, and a gratuity of fourteen days' pay for every three months' additional service. A short experience of these terms proved they were not sufficiently attractive to men who were losing time in starting in private practice. In December, 1901, it was approved that the increase of 5/- a day should be given at twelve months' service, and an additional 2/6 after eighteen months' service. These improved terms did not lessen the difficulties, so that in May, 1902, it was decided to offer £1 a day pay for the first year, to be raised to 25/- a day after twelve months' service, and 27/6 after eighteen months' service. How far these terms would have worked had the war continued we cannot say, but £1 a day, and the usual allowances and gratuity, should be the minimum offered to a civilian service which has no claim on the State in the event of an injury, other than that received in action, or impairment of health the result of service in the Field.

The total number of Civil Veterinary Surgeons engaged for the war was 283, of whom seven died during the campaign, and one or more shortly afterwards. The first arrivals occurred at the end of 1899; by the time the war had lasted six months there were 120 civil veterinary surgeons in the Field. The difficulties and disadvantages under which these gentlemen laboured are fully set forth at p. 28a; rank was withheld from them (see p. 48d), though granted to Yeomanry and Colonial veterinary officers, and, in fact, to everyone in the Field excepting Civil Surgeons and Civil Veterinary Surgeons. They possessed no authority (p. 27d), though frequently placed in positions where such was essential to the working of their charge (see pp. 73b and 197d). It is unnecessary to reiterate their difficulties, they were many and acute. In one respect the regular service failed towards them, and that was in not making sufficient allowance for their ignorance of military matters. We forgot, in the stress of events, the years it had taken us to acquire a working knowledge of the military machine, and often, we fear, expected more than the average civilian could yield. That there were many weak vessels among them goes without saying where no selection was possible, but many of their difficulties would have disappeared had there been administrative officers to whom they could have looked for instruction, instead of being—to use a local expression—"dumped down" on the veldt and left to their own devices. When it is remembered that the average proportion of Civil Veterinary Surgeons to Regular officers was as $4\frac{1}{2}$ to one, it will be seen what a large share of the veterinary duties they were destined to bear.

The Civil Veterinary Service when trained did useful service; some of its members possessed marked ability and did excellent work, as the pages of this History have testified. It took six months to train a first-rate man, the average man took a year, and many were never trained (see p. 28c). This was due in many cases to ineptitude, in others to lack of opportunity, owing to the insignificant number of administrative officers.

The mushroom-like character of the forces used in the war had its reflex in the veterinary service. Regiments were created and disbanded in the course of a few months, and new regiments took their place. Similarly, civil veterinary surgeons, just as they were getting in touch with their work suddenly returned home on expiration of contract, and the whole business of acquiring knowledge by the process of blundering had to be gone through with the new arrivals. Such a system is hopeless as an effective organization. This system of civil veterinary aid in war is absolutely indefensible. Over and over again we have insisted that the Field is not the place to carry out a primary training, no matter for what service the person is intended.

THE SEA-TRANSPORT VETERINARY SERVICE.

This has already been dealt with. We have seen, at p. 255b, that the work of the British Veterinary Surgeons gave satisfactory results, but the foreigners engaged by the Remount Department were badly reported upon, and brought great discredit on the British service. The rates of remuneration are dealt with at p. 262b. The difficulties of Transport Veterinary Surgeons at sea are fully recorded at p. 255c *et seq*, and their equally great difficulties ashore have been illustrated in detail at p. 262d, footnote *et seq*.

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